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TOTAL FOR ALL FILES
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L77 ANSWER 1 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2005:22946 USPATFULL
TITLE: Layered aligned polymer structures and methods of making same
INVENTOR(S): Braithwaite, Gavin J. C., Cambridge, MA, UNITED STATES
Ruberti, Jeffrey W., Lexington, MA, UNITED STATES
PATENT ASSIGNEE(S): Cambridge Polymer Group, Inc., Boston, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005019488	A1	20050127
APPLICATION INFO.:	US 2003-611674	A1	20030630 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-306825, filed on 27 Nov 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-337286P	20011130 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Monica Grewal, Esq., BOWDITCH & DEWEY, LLP, 161 Worcester Road, P.O. Box 9320, Framingham, MA, 01701-9320
NUMBER OF CLAIMS: 90
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 25 Drawing Page(s)
LINE COUNT: 2189
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention includes a method of producing a nanostructured artificial template comprising more than one thin, oriented layer of polymer material. The material is preferably produced by the method of introducing a shearing flow to a free surface in a predominantly monomeric solution of the self-assembling polymer sub-units, and inducing polymerization or growth of the monomer while in this shearing flow. The system for forming the oriented layer of material provides relative movement between a delivery system and the substrate on or over which the material is deposited. The rate of flow of the material from the delivery system and the relative velocity between the deposition surface and the material as it is delivered to the surface are controlled to properly orient the material at the desired thickness. These rates can be adjusted to vary the properties of the film in a controlled manner. Preferred embodiments include either angular or linear relative movement between the delivery system and the substrate. The nanostructured artificial template is useful for inducing the production of a templated extracellular matrix by a population of cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L77 ANSWER 2 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2004:44503 USPATFULL
TITLE: Methods of diagnosis of angiogenesis, compositions and methods of screening for angiogenesis modulators
INVENTOR(S): Murray, Richard, Cupertino, CA, UNITED STATES

Glynne, Richard, Palo Alto, CA, UNITED STATES
Watson, Susan R., El Cerrito, CA, UNITED STATES
Aziz, Natasha, Palo Alto, CA, UNITED STATES
PATENT ASSIGNEE(S): Eos Biotechnology, Inc., South San Francisco, CA, UNITED STATES, 94080 (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004033495	A1	20040219	
APPLICATION INFO.:	US 2002-211462	A1	20020801	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-310025P	20010803 (60)
	US 2001-334244P	20011129 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
LINE COUNT: 24599
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described herein are methods and compositions that can be used for diagnosis and treatment of angiogenic phenotypes and angiogenesis-associated diseases. Also described herein are methods that can be used to identify modulators of angiogenesis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L77 ANSWER 3 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2003:271112 USPATFULL
TITLE: Novel proteins and nucleic acids encoding same
INVENTOR(S): Grosse, William M., Branford, CT, UNITED STATES
Alsobrook, John P., II, Madison, CT, UNITED STATES
Lepley, Denise M., Branford, CT, UNITED STATES
Burgess, Catherine E., Wethersfield, CT, UNITED STATES
Mishra, Vishnu, Gainesville, FL, UNITED STATES
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Shimkets, Richard A., West Haven, CT, UNITED STATES
Zerhusen, Bryan D., Branford, CT, UNITED STATES
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Gerlach, Valerie, Branford, CT, UNITED STATES
MacDougall, John R., Hamden, CT, UNITED STATES
Millet, Isabelle, Milford, CT, UNITED STATES
Stone, David J., Guilford, CT, UNITED STATES
Gunther, Erik, Branford, CT, UNITED STATES
Ellerman, Karen, Branford, CT, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003190715	A1	20031009	
APPLICATION INFO.:	US 2001-976782	A1	20011012	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-240113P	20001012 (60)
	US 2000-240662P	20001016 (60)
	US 2000-240732P	20001016 (60)
	US 2000-240625P	20001016 (60)
	US 2000-240648P	20001016 (60)
	US 2000-240703P	20001016 (60)
	US 2000-241190P	20001016 (60)

	US 2000-240637P	20001016 (60)
	US 2000-240669P	20001016 (60)
	US 2001-262455P	20010118 (60)
DOCUMENT TYPE:	Utility	
FILE SEGMENT:	APPLICATION	
LEGAL REPRESENTATIVE:	Ivor R. Elrifi, Mintz, Levin, Cohn, Ferris,, Glovsky and Popeo, P.C., One Financial Center, Boston, MA, 02111	
NUMBER OF CLAIMS:	49	
EXEMPLARY CLAIM:	1	
LINE COUNT:	9839	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed herein are nucleic acid sequences that encode novel polypeptides. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivatives, variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L77 ANSWER 4 OF 10 USPATFULL on STN

ACCESSION NUMBER:	2003:205269 USPATFULL		
TITLE:	Layered aligned polymer structures and methods of making same		
INVENTOR(S):	Braithwaite, Gavin J. C., Cambridge, MA, UNITED STATES Ruberti, Jeffrey W., Lexington, MA, UNITED STATES		
PATENT ASSIGNEE(S):	Cambridge Polymer Group, Inc., Boston, MA (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003141618	A1	20030731
APPLICATION INFO.:	US 2002-306825	A1	20021127 (10)
	NUMBER		DATE
PRIORITY INFORMATION:	US 2001-337286P		20011130 (60)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		
LEGAL REPRESENTATIVE:	MONICA GREWAL, ESQ., BOWDITCH & DEWEY, LLP, 161 Worcester Road, P.O. Box 9320, Framingham, MA, 01701-9320		
NUMBER OF CLAIMS:	79		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	18 Drawing Page(s)		
LINE COUNT:	1276		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention includes a method of producing a thin, oriented layer of polymer material. The material is preferably produced by the method of introducing a shearing flow to a free surface in a predominantly monomeric solution of the self-assembling polymer sub-units, and inducing polymerization or growth of the monomer while in this shearing flow. The system for forming the oriented layer of material provides relative movement between a delivery system and the substrate on or over which the material is deposited. The rate of flow of the material from the delivery system and the relative velocity between the deposition surface and the material as it is delivered to the surface are controlled to properly orient the material at the desired thickness. These rates can be adjusted to vary the properties of the film in a controlled manner. Preferred embodiments include either angular or linear relative movement between the delivery system and the substrate.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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DUPLICATE 1
ACCESSION NUMBER: 2003:363390 BIOSIS
DOCUMENT NUMBER: PREV200300363390
TITLE: Keratocan-deficient mice display alterations in corneal structure.
AUTHOR(S): Liu, Chia-Yang [Reprint Author]; Birk, David E.; Hassell, John R.; Kane, Bradley; Kao, Winston W.-Y.
CORPORATE SOURCE: Bascom Palmer Eye Inst., Dept. of Ophthalmology, McKnight Vision Research Center, University of Miami School of Medicine, 1638 N. W. 10th Ave., Rm. 621, Miami, FL, 33136, USA
cliu2@med.miami.edu
SOURCE: Journal of Biological Chemistry, (June 13 2003) Vol. 278, No. 24, pp. 21672-21677. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Aug 2003
Last Updated on STN: 6 Aug 2003

AB Keratocan (Kera) is a cornea-specific keratan sulfate proteoglycan (KSPG) in the adult vertebrate eye. It belongs to the small leucine-rich proteoglycan (SLRP) gene family and is one of the major components of extracellular KSPG in the vertebrate corneal stroma. The Kera gene is expressed in ocular surface tissues including cornea and eyelids during morphogenesis. Corneal KSPGs play a pivotal role in matrix assembly, which is accountable for corneal transparency. In humans, mutations of the KERA gene are associated with cornea plana (CNA2) that manifests decreases in vision acuity due to the flattened forward convex curvature of cornea. To investigate the biological role of the Kera gene and to establish an animal model for corneal plana, we generated Kera knockout mice via gene targeting. Northern and Western blotting and immunohistochemical analysis showed that no Kera mRNA or keratocan protein was detected in the Kera-/- cornea. The expression levels of other SLRP members including lumican, **decorin**, and fibromodulin were not altered in the Kera-/- cornea as compared with that of the wild-type littermates. Mice lacking keratocan have normal corneal transparency at the age of 12 months. However, they have a thinner corneal stroma and a narrower cornea-iris angle of the anterior segment in comparison to the wild-type littermates. As demonstrated by transmission electron microscopy, Kera-/- mice have larger stromal fibril diameters and less organized packing of collagen fibrils in stroma than those of wild type. Taken together, our results showed that ablation of the Kera gene resulted in subtle structural alterations of collagenous matrix and did not perturb the expression of other SLRPs in cornea. Keratocan thus plays a unique role in maintaining the appropriate **corneal shape** to ensure normal vision.

L77 ANSWER 6 OF 10 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:477606 SCISEARCH
THE GENUINE ARTICLE: 557VA
TITLE: Altered collagen fibril formation in the sclera of lumican-deficient mice
AUTHOR: Austin B A; Coulon C; Liu C Y; Kao W W Y; Rada J A (Reprint)
CORPORATE SOURCE: Univ N Dakota, Sch Med & Hlth Sci, Dept Anat & Cell Biol, 501 N Columbia Rd, Grand Forks, ND 58202 USA (Reprint); Univ N Dakota, Sch Med & Hlth Sci, Dept Anat & Cell Biol, Grand Forks, ND 58202 USA; GAIA Grp, Novato, CA USA; Univ Cincinnati, Med Ctr, Dept Ophthalmol, Cincinnati, OH 45267 USA
COUNTRY OF AUTHOR: USA
SOURCE: INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCIENCE, (JUN 2002) Vol. 43, No. 6, pp. 1695-1701.

Publisher: ASSOC RESEARCH VISION OPHTHALMOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA.
ISSN: 0146-0404.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 38
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB PURPOSE. To better understand the role of lumican (**corneal** keratan sulfate proteoglycan) in the scleral extracellular matrix, collagen fibril size, **shape**, and organization were evaluated in the sclera of wild-type mice and in mice homozygous or heterozygous for a null mutation in the lumican gene.

METHODS. Anterior and posterior sclera from 6-month-old wild-type (lum(+)/lum(+)) and lumican-deficient mice (lum(+)/lum(-) and lum(-)/lum(-)) were analyzed by transmission electron microscopy. In addition, lumican was characterized in the sclera of wild-type and lumican-deficient mice by Western blot analyses.

RESULTS. Lumican was present in the mouse sclera as an approximately 48-kDa core protein containing short glycosaminoglycan side chains consisting of moderate- to low-sulfated keratan sulfate. The wild-type mouse sclera consisted of irregularly arranged lamellae of collagen fibrils with an average diameter of 47.37 +/- 0.648 nm in the anterior sclera and 54.68 +/- 0.342 nm the posterior sclera. Collagen fibrils in the sclera of lumican mutant mice (lum(+)/lum(-) and lum(-)/lum(-)) were significantly larger in diameter in anterior (72.61 +/- 0.445 and 84.47 +/- 0.394 nm, respectively) and posterior (75.92 +/- 0.361 and 80.90 +/- 0.490 nm, respectively) scleral regions compared with wild-type mice ($P < 0.001$).

CONCLUSIONS. The results of the present study indicate that null mutations in one or both alleles of the lumican gene result in significant defects in scleral collagen fibril formation that could lead to alterations in ocular shape and size and severely affect vision.

L77 ANSWER 7 OF 10 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 2

ACCESSION NUMBER: 2002:339684 BIOSIS
DOCUMENT NUMBER: PREV200200339684
TITLE: Electron microscopic and immunohistochemical examination of scarred human cornea re-treated by excimer laser.
AUTHOR(S): Bleckmann, Heinrich [Reprint author]; Schnoy, Norbert; Kresse, Hans
CORPORATE SOURCE: Augenabteilung der Schlosspark-Klinik Berlin, Heubnerweg 2, 14059, Berlin, Germany
Prof.Dr.H.Bleckmann@t-online.de
SOURCE: Graefe's Archive for Clinical and Experimental Ophthalmology, (April, 2002) Vol. 240, No. 4, pp. 271-278. print.
CODEN: GACODL. ISSN: 0721-832X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jun 2002
Last Updated on STN: 12 Jun 2002

AB Purpose: To elucidate differences, at the macromolecular level, in corneal tissue subjected to repeated argon fluoride excimer treatment. Methods: A light microscopic, electron microscopic, and immunohistochemical study was performed on a scarred human cornea. Results: Keratocytes were enlarged with an expanded endoplasmic reticulum and exhibited a fibroblastic appearance. Amorphous material was observed extracellularly. Collagen fibrils exhibited a disordered arrangement while banding patterns of diameter were normal. Immunohistochemical investigation of several collagen types, of collagen-associated proteoglycans, and of basement membrane components demonstrated an enhanced immunoreactivity of all of them in the scarred area. Type V collagen was found as a normal component of the epithelial basement membrane whereas types I and III collagen were present beneath Bowman's layer. Excimer-laser-treated sections revealed considerably stronger subepithelial staining for collagen types I, III,

IV, and V. Laminin-1, a typical component of basement membranes, was detectable throughout the scarred tissue. The small proteoglycans **decorin** and fibromodulin accumulated in a patch-like manner in the scarred tissue below the epithelium, whereas biglycan was expressed by the epithelium and throughout the stroma. Lumican was expressed most strongly by the epithelium and rather equally distributed in the excimer-laser-treated and in the normal stroma. Conclusion: Effects of argon laser treatment of the cornea must be regarded as a process acting over many months. Intra- and extracellular structures and components are involved and influence the unpredictable **shape** of the **corneal** architecture.

L77 ANSWER 8 OF 10 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 3

ACCESSION NUMBER: 2002360973 EMBASE
TITLE: Mice deficient in small leucine-rich proteoglycans: Novel in vivo models for osteoporosis, osteoarthritis, Ehlers-Danlos syndrome, muscular dystrophy, and corneal diseases.
AUTHOR: Ameye L.; Young M.F.
CORPORATE SOURCE: M.F. Young, Craniofacial/Skeletal Dis. Branch, NIDCR, NIH, Building 30, Bethesda, MD 20892, United States. myoung@dir.nidcr.nih.gov
SOURCE: Glycobiology, (1 Sep 2002) 12/9 (107R-116R). Refs: 100 ISSN: 0959-6658 CODEN: GLYCE3
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 012 Ophthalmology
020 Gerontology and Geriatrics
029 Clinical Biochemistry
031 Arthritis and Rheumatism
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Small leucine-rich proteoglycans (SLRPs) are extracellular molecules that bind to TGFβs and collagens and other matrix molecules. In vitro, SLRPs were shown to regulate collagen fibrillogenesis, a process essential in development, tissue repair, and metastasis. To better understand their functions in vivo, mice deficient in one or two of the four most prominent and widely expressed SLRPs (biglycan, **decorin**, fibromodulin, and lumican) were recently generated. All four SLRP deficiencies result in the formation of abnormal collagen fibrils. Taken together, the collagen phenotypes demonstrate a cooperative, sequential, timely orchestrated action of the SLRPs that altogether **shape** the architecture and mechanical properties of the collagen matrix. In addition, SLRP-deficient mice develop a wide array of diseases (osteoporosis, osteoarthritis, muscular dystrophy, Ehlers-Danlos syndrome, and **corneal** diseases), most of them resulting primarily from an abnormal collagen fibrillogenesis. The development of these diseases by SLRP-deficient mice suggests that mutations in SLRPs may be part of undiagnosed predisposing genetic factors for these diseases. Although the distinct phenotypes developed by the different singly deficient mice point to distinct in vivo function for each SLRP, the analysis of the double-deficient mice also demonstrates the existence of rescuing/ compensation mechanisms, indicating some functional overlap within the SLRP family.

L77 ANSWER 9 OF 10 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2002232352 ESBIOBASE
TITLE: Mice deficient in small leucine-rich proteoglycans: Novel in vivo models for osteoporosis, osteoarthritis, Ehlers-Danlos syndrome, muscular dystrophy, and corneal diseases
AUTHOR: Ameye L.; Young M.F.
CORPORATE SOURCE: M.F. Young, Craniofacial/Skeletal Dis. Branch, NIDCR, NIH, Building 30, Bethesda, MD 20892, United States.

E-mail: myoung@dir.nidcr.nih.gov
SOURCE: Glycobiology, (01 SEP 2002), 12/9 (107R-116R), 100 reference(s)
CODEN: GLYCE3 ISSN: 0959-6658
DOCUMENT TYPE: Journal; (Short Survey)
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Small leucine-rich proteoglycans (SLRPs) are extracellular molecules that bind to TGFβs and collagens and other matrix molecules. In vitro, SLRPs were shown to regulate collagen fibrillogenesis, a process essential in development, tissue repair, and metastasis. To better understand their functions in vivo, mice deficient in one or two of the four most prominent and widely expressed SLRPs (biglycan, **decorin**, fibromodulin, and lumican) were recently generated. All four SLRP deficiencies result in the formation of abnormal collagen fibrils. Taken together, the collagen phenotypes demonstrate a cooperative, sequential, timely orchestrated action of the SLRPs that altogether **shape** the architecture and mechanical properties of the collagen matrix. In addition, SLRP-deficient mice develop a wide array of diseases (osteoporosis, osteoarthritis, muscular dystrophy, Ehlers-Danlos syndrome, and **corneal** diseases), most of them resulting primarily from an abnormal collagen fibrillogenesis. The development of these diseases by SLRP-deficient mice suggests that mutations in SLRPs may be part of undiagnosed predisposing genetic factors for these diseases. Although the distinct phenotypes developed by the different singly deficient mice point to distinct in vivo function for each SLRP, the analysis of the double-deficient mice also demonstrates the existence of rescuing/ compensation mechanisms, indicating some functional overlap within the SLRP family.

L77 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2001:208121 CAPLUS
DOCUMENT NUMBER: 134:242743
TITLE: Composition for stabilizing corneal tissue during or after **orthokeratology** lens wear
INVENTOR(S): Dewoolfson, Bruce H.; Devore, Dale P.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2001019386	A2	20010322	WO 2000-US25190		20000915
WO 2001019386	A3	20010927			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
AU 2000075802	A5	20010417	AU 2000-75802		20000915
EP 1218027	A2	20020703	EP 2000-965007		20000915
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL					
JP 2003509376	T2	20030311	JP 2001-523018		20000915
PRIORITY APPLN. INFO.:			US 1999-153959P	P	19990915
			US 1999-173801P	P	19991230
			WO 2000-US25190	W	20000915

AB Two types of compns. having an eye-drop delivery system are used during or after an **orthokeratol**. procedure to prevent or retard relaxation of corneal tissue back to the original anterior curvature of the cornea. Each composition functions independently from the others and is a different approach of preparing a stabilizing agent. The first composition is directed to a biol. compatible composition comprising fibril associated collagens with interrupted triple helixes (FACITs) and/or small leucine-rich repeat proteoglycans (SLRPs). The fibril associated collagen family includes various types of collagens, such as type VI, type XX, type XII, and type XIV. The small leucine-rich repeat proteoglycans family includes **decorin**, keratocan, biglycan, epiphycan, lumican, mimecan, and fibromodulin. The second composition includes the enzyme found as a normal component of tissues, plasma, or epidermis, such as **transglutaminase**.

=>

Detail Page

2.Document ID: WO0119386A3

Application Number: 25190

Publication Date: 99990101
Title:

- COMPOSITION FOR STABILIZING CORNEAL TISSUE DURING OR AFTER ORTHOKERATOLOGY LENS WEAR
- COMPOSITION DE STABILISATION DU TISSU DE LA CORNEE PENDANT OU APRES LE PORT DE LENTILLE D'ORTHOKERATOLOGIE

Inventor(s):

- DEVORE DALE P
- DEWOOLFSON BRUCE H

Assignee:

- DEVORE DALE P
- DEWOOLFSON BRUCE H

Priority:

- Priority Country: US
- Priority Number: 15395999
- Priority Date: 19990915

Priority:

- Priority Country: US
- Priority Number: 17380199
- Priority Date: 19991230

IPC:

- A61K 38/39
- A61K 38/17
- A61K 38/45
- A61P 27/02

WEST Search History

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DATE: Thursday, March 03, 2005

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	DB=PGPB,USPT,USOC; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L5	(cornea$2 with shape? or orthokeratolog$4) and ((type VI with collage) or decorin or transglutaminase)	2
☐	L4	(cornea$2 with shape? or orthokeratolog$4) and transglutaminase	2
☐	L3	(cornea$2 with shape?) andtransglutaminase	0
☐	L2	(cornea$2 with shape?) same transglutaminase	0
☐	L1	orthokeratolog$4 same transglutaminase	0

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 2 of 2 returned.

1. Document ID: US 20030157073 A1

Using default format because multiple data bases are involved.

L4: Entry 1 of 2 File: PGPB Aug 21, 2003

PGPUB-DOCUMENT-NUMBER: 20030157073
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030157073 A1

TITLE: Methods for pretreating a subject with apoptotic cells

PUBLICATION-DATE: August 21, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Peritt, David L.	Bala Cynwyd	PA	US	
Harriman, Gregory	Paoli	PA	US	

US-CL-CURRENT: 424/93.21; 424/93.7

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

2. Document ID: US 20030139466 A1

L4: Entry 2 of 2 File: PGPB Jul 24, 2003

PGPUB-DOCUMENT-NUMBER: 20030139466
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030139466 A1

TITLE: Methods for pretreating a subject with extracorporeal photopheresis

PUBLICATION-DATE: July 24, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Peritt, David L.	Bala Cynwyd	PA	US	
Harriman, Gregory	Paoli	PA	US	

US-CL-CURRENT: 514/453

ABSTRACT:

The present invention relates to methods for treating a subject predisposed to an autoimmune

disease with extracorporeal photopheresis or an effective amount of apoptotic cells before the clinical manifestation of a symptom associated with the autoimmune disease. The present invention alsorelates to methods for treating a subject predisposed to an atopic disease with extracorporeal photopheresis or an effective amount of apoptotic cells before the clinical manitfestation of a symptom associated with the atopic disease. The present invention further relates to methods for treating a transplant donor and/or a transplant recipient, or an implant recipient with extracorporeal photopheresis or an effective amount of apoptotic cells prior to the transplant or implantation procedure.

Term	Documents
TRANSGLUTAMINASE	1585
TRANSGLUTAMINASES	422
CORNEA$2	0
CORNEA	12866
CORNEAE	205
CORNEAI	1
CORNEAIL	2
CORNEAL	12541
CORNEALE	1
CORNEALK	1
CORNEALS	2
((CORNEA$2 WITH SHAPE? OR ORTHOKERATOLOG$4) AND TRANSGLUTAMINASE).PGPB,USPT,USOC.	2

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